

can be difficult to achieve as medicine becomes more complex, fragmented, episodic, and impersonal.

Understanding medical altruism is also likely to be important in workforce planning particularly if, as in the UK National Health Service, recruitment and retention of medical and nursing staff are problematic. It may well be that the conditions that encourage clinicians to join and stay in their posts are not dissimilar to those that are needed for the development of altruistic behaviours. If it is also true that the maintenance of these behaviours depends on the recognition of individuals with similar characteristics—clinical and professional values—and on the expectation of reciprocity, then there is a strong message here for managers and policy makers. Disenfranchisement and disengagement are dimensions of demoralisation and burnout, a constant threat to physicians' health.⁹ Workforce planning needs to be more than a numbers game and must pay explicit attention to the working

conditions, incentives, and rewards provided for all healthcare workers.

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Epidemic of cardiovascular disease in South Asians

Prevention must start in childhood

Papers p 635

People with ancestry in the countries of the Indian subcontinent (South Asians), comprising more than one fifth of the global population, are highly susceptible to cardiovascular diseases. This susceptibility is well demonstrated in South Asian migrants in places as diverse as the United Kingdom, South Africa, the Caribbean, Singapore, the United States, Canada, and urban India.¹ Unless controlled, this epidemic, which is starting in urban settings but spreading rapidly to semi-urban and rural settings, will thwart global control of cardiovascular diseases.² Research on several communities of the South Asian diaspora has provided insights that are vital to the control of cardiovascular diseases in South Asians worldwide. In this issue Whincup et al extend the strong tradition of British research by reporting observations in children (p 635).³

In 1994 Whincup et al measured insulin, glucose, and other biochemical risk factors and made social and anthropometric observations in 8-11 year olds, mostly Pakistani Muslims, in 10 British towns. Their work primarily contributes to two questions. Firstly, what is the potential role of insulin resistance in explaining the high risk of cardiovascular diseases in South Asians. Secondly, given a particular exposure to a risk factor, might South Asians be at higher risk or more susceptible to its effects?

The causes of the high cardiovascular risk in South Asians, other than artefacts of data collection (which probably play some part),⁴ can be considered under four categories: excess exposure to known risk factors, greater susceptibility, new risk factors, or competing causes.

Excess exposure to risk factors—The explanation that South Asians are more exposed to the causal risk factors is usually dismissed,¹ possibly too readily. Except for smoking, which is lower in Indian (but not Pakistani and Bangladeshi) men and all South Asian women, the pattern is complex.⁵⁻⁷ The established risk factors com-

moner in South Asians include low high density lipoprotein cholesterol, diabetes (much commoner in South Asians), and lack of aerobic exercise. When the risk profile is seen in the context of social factors linked to coronary heart disease such as relative poverty, social upheaval after migration, and long working hours, this explanation deserves more consideration. Whincup et al showed that parents of South Asian children were much more likely to be in manual occupations, and on a broad range of biological risk factors the children are either similar to or worse off than the Europeans.

Greater susceptibility—The explanation that South Asians are more susceptible to established risk factors for coronary heart disease risk has not been systematically studied. Proposed mechanisms include genetic differences (unidentified) or a mismatch between fetal and early life metabolism and that in middle age. A third possibility is that rapid change in some risk factors may itself confer a risk. Punjabis in Southall had a mean serum cholesterol of 6.5 mmol/l compared with 4.9 mmol/l for their siblings in Punjab, India.⁸ Rapid rises, as implied here, may confer a risk beyond that predicted by a static measure. Whincup et al's observations contribute to this explanation.³ They have shown what some have long suspected: that the relation between risk factor and outcome may differ by ethnic group. South Asians may indeed be more susceptible—as shown in Whincup et al's data by a comparatively steeper slope in the regression line describing the association between risk factor (here obesity) and outcome (here insulin). Patel et al showed that conventional indicators of obesity were inconsistently associated with components of the metabolic syndrome in Chinese, South Asian, and European populations, with demonstrable interactions between indicators such as waist measurement, waist-hip ratio, and ethnicity.⁹ Whincup et al's findings might be

explained by the greater tendency to central deposition of fat in South Asian children.¹⁰ Important observations, such as those of Whincup et al and Patel et al, made on cross sectional data, need to be verified in cohort studies. But none of the many cardiovascular cohort studies in the United Kingdom can yield risk-outcome data by ethnic group.⁴

New risk factors—The third explanation is that specific risk factors, not yet established or discovered, may explain high risk. The search for a specific cause has led to many hypotheses, including the use of ghee and other cooking oils, subclinical hypothyroidism, central obesity, stress, racism, insulin resistance, a thrifty genotype, a thrifty phenotype, low vitamin C, high homocysteine, endothelial dysfunction, high levels of lipoprotein a, and other specific lipid abnormalities. No “South Asian cause” of coronary heart disease has been proved, though each new idea has diverted attention from established risk factors. The best studied hypothesis is that the high prevalence of insulin resistance, independent of diabetes, underlies the high rates of coronary heart disease in South Asians.¹ Rigorous tests of this hypothesis, based on prospective studies, are awaited, but Whincup et al provide data of interest on children. Though South Asian children were no more obese than those of European origin, fasting and 30 minute post load insulin were about 50% higher.

Competing causes—The fourth, rarely considered explanation, is that there are fewer competing causes of death in middle aged South Asians, particularly as cancer rates are comparatively low. Whincup et al do not touch on this concept.

Whincup et al have paved the way to paying more attention to young South Asians, mostly born in the United Kingdom. They show that if insulin and insulin resistance do turn out to be causally related to coronary heart disease in South Asians then preventive action will need to take place early. Simmons reported from New Zealand that Indian babies had less insulin in cord blood than European, Maori, and Pacific Islander babies.¹¹ Further studies are needed to corroborate these findings; to confirm that findings in Pakistanis apply to other South Asian groups—as is likely; and to establish exactly when the tendency to insulin resistance emerges and why. Even if insulin

resistance is not directly causative of coronary heart disease, it is predictive of diabetes, a key and highly prevalent risk factor in South Asians. This work emphasises that the prevention of diabetes must start in early life.

This study has policy and service implications. South Asians’ poor knowledge and understanding of coronary heart disease and diabetes are shocking, particularly in Bangladeshis and Pakistanis.¹² In addition to conveying effective and accurate messages about coronary heart disease prevention in adults we must weave in the key message that children are at risk. As all the established risk factors are important in South Asians, the health promotion challenge is formidable.

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Time to abandon the “tendinitis” myth

Painful, overuse tendon conditions have a non-inflammatory pathology

Tendinitis such as that of the Achilles, lateral elbow, and rotator cuff tendons is a common presentation to family practitioners and various medical specialists.¹ Most currently practising general practitioners were taught, and many still believe, that patients who present with overuse tendinitis have a largely inflammatory condition and will benefit from anti-inflammatory medication. Unfortunately this dogma is deeply entrenched. Ten of 11 readily available sports medicine texts specifically recommend non-steroidal anti-inflammatory drugs for treating painful conditions like Achilles and patellar tendinitis

despite the lack of a biological rationale or clinical evidence for this approach.^{2 3}

Instead of adhering to the myths above, physicians should acknowledge that painful overuse tendon conditions have a non-inflammatory pathology. Light microscopy of patients operated on for tendon pain reveals collagen separation⁴⁻⁶—thin, frayed, and fragile tendon fibrils, separated from each other lengthwise and disrupted in cross section. There is an apparent increase in tenocytes with myofibroblastic differentiation (tendon repair cells) and classic inflammatory cells are usually absent.⁴ This is tendinosis and it was first

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